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Set Items Description
S1 140254 PARTICULATE?? OR (ALUMINUM(W) HYDROXIDE) OR (COLLOIDAL(W) GO-
LD) OR (POLYSTYRENE(W) LATEX)
S2 296739 AGGREGAT?
S3 3984 S1 AND S2
S4 288154 SUGAR OR TREHALOSE OR CARBOHYDRATE
S5 75 S3 AND S4
S6 57 RD (unique items)
S7 25 S6 AND PY<=1994
S8 4715057 PREVENT? OR REDUC?
S9 7 S7 AND S8
? s scavenger(5n) radical
30016 SCAVENGER
359852 RADICAL
S10 9049 SCAVENGER (5N) RADICAL
? s s4 and s10
288154 S4
9049 S10
S11 100 S4 AND S10
? s mannitol
S12 30088 MANNITOL
? s s12 and s10
30088 S12
9049 S10
S13 558 S12 AND S10
? s s13 and py<=1994
Processing
558 S13
25468139 PY<=1994
S14 201 S13 AND PY<=1994
? s s14 and s1
201 S14
140254 S1
S15 1 S14 AND S1
? t s15/3,k,ab/1

15/3,K,AB/1 (Item 1 from file: 34)
DIALOG(R) File 34:SciSearch(R) Cited Ref Sci
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03315480 Genuine Article#: NW488 Number of References: 44
Title: EFFECT OF SCAVENGERS OF ACTIVE OXYGEN SPECIES ON CELL-DAMAGE CAUSED
IN CHO-K1 CELLS BY PHENYLHYDROQUINONE, AN O-PHENYLPHENOL METABOLITE (Abstract Available)

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Abstract: Phenylhydroquinone (PHQ), a metabolite of o-phenylphenol (OPP), is easily autoxidized to phenylbenzoquinone (PBQ) via the semiquinone (phenylsemiquinone, PSQ) with concomitant production of superoxide anion radicals (O-2(radical-anion)). We have used scavengers of active oxygen species to examine whether or not O-2(radical-anion) produced during oxidation of PHQ is related to cell damage in CHO-K1 cells. PHQ at 10 μ g/ml (3-h treatment) induced sister-chromatid exchange (SCE), endoreduplication (ERD) and cell-cycle delay in CHO-K1 cells. These effects were inhibited by catalase (280 U/ml), a scavenger of hydrogen peroxide (H2O2), as well as by the reductants, ascorbate (3 mM) and GSH (1 mM). Mannitol (50 mM), a scavenger of hydroxyl radical (OH.), was ineffective and superoxide dismutase (SOD, 150

U/ml), a **scavenger** of O-2(radical-anion), or SOD plus catalase rather intensified the toxicity as did aminotriazole (20 mM), an inhibitor of catalase. Analyses of incubation solutions by HPLC showed that the extent of cell damage is correlated with PHQ loss; catalase suppressed PHQ loss, whereas SOD promoted it. The correlation was more clearly seen in the time courses of cell death and PHQ loss during incubation of PHQ with each of the scavengers of active oxygen species. These results show that neither O-2(radical-anion) nor OH. participates in the cell damage, but rather H2O2 generated via dismutation of O-2(radical-anion) may participate, probably by accelerating the autoxidation of PHQ and thus causing an increase in the production of toxic intermediates. In fact, conversion of PHQ to PBQ, a reactive product, was demonstrated during incubation with PHQ in phosphate-buffered saline by following the changes in W-visible spectra of PHQ. Inclusion of H2O2 (0.2 or 1 mM) in the incubation mixture accelerated the PHQ loss. The present results can be explained in terms of the autoxidation mechanism of hydroquinone proposed by O'Brien (1991). Different from the results in the absence of S9 mix, the cell damage induced by 50 μ g/ml OPP in the presence of S9 mix was not influenced by any of the scavengers of active oxygen species used. We conclude that PHQ causes cytotoxic and genotoxic effects through its autoxidation, both enzymatic and nonenzymatic, and that reactive intermediate(s) such as PSQ and/or PBQ may be ultimately responsible for the effects. H2O2 formed during the oxidation process participates in the damaging effects caused in the absence of S9 mix, probably by accelerating the autoxidation.

, 1994

...Abstract: peroxide (H2O2), as well as by the reductants, ascorbate (3 mM) and GSH (1 mM). Mannitol (50 mM), a **scavenger** of hydroxyl radical (OH.), was ineffective and superoxide dismutase (SOD, 150 U/ml), a **scavenger** of O-2(radical-anion), or SOD plus catalase rather intensified the toxicity as did aminotriazole (20 mM), an...

Research Fronts: 92-0790 001 (BIOASSAY-DIRECTED CHEMICAL-ANALYSIS OF AMBIENT AIR PARTICULATE EXTRACTS; SALMONELLA MUTAGENICITY; ATMOSPHERIC POLYCYCLIC AROMATIC-HYDROCARBONS; AMES TEST)

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